August 1965

U.S. FOREST SERVICE
RESEARCH NOTE RM - 50

The Mechanism of Fenuron Injury to Plants[1]

Edwin A. Davis[2]

Fenuron (3-phenyl-1,1-dimethylurea), like other substituted phenylurea herbicides, is absorbed by plant roots and is transported upwards to the leaves where its major toxic action occurs (Minshall 1954, Muzik et al., 1954).[3] The substituted phenylurea herbicides are potent inhibitors of photosynthesis that block a reaction concerned with the production of oxygen (Bishop 1958, Cooke 1956, Spikes 1956, Wessels and van der Veen 1956). It has been proposed that the toxic symptoms produced by these chemicals are primarily a result of the lack of photosynthate brought about by the action of the chemicals (Gentner and Hilton 1960). It is true that in the absence of photosynthesis, starvation would result with the accompanying development of visible injury. But a blocked photosynthetic reaction might also be a direct rather than an indirect cause of injury.

The acorn of shrub live oak constitutes a sizable reservoir of foodstuff for the developing seedling. Consequently, if the hypothesis that visible leaf injury is the direct result of starvation is correct, young seedlings should not become visibly injured until the reserves in the acorns are exhausted. Bathing the acorns in glucose solution should then postpone starvation and further delay the onset of injury symptoms. Also, it would be expected that seedlings whose acorns had been excised would be injured soon after treatment, whereas seedlings with intact acorns would not develop injury symptoms until the acorn reserves were exhausted.

One objective of the experiments reported here was to evaluate the hypothesis that visible leaf injury from fenuron treatments is due directly to starvation. Additional objectives were to determine the influence of fenuron on root growth of shrub live oak seedlings, and to relate root growth to shoot injury.

Materials and Methods

Large acorns, collected from a single tree, were germinated on moist vermiculite. The acorns measured about 2.4 cm. long and 1.3 cm. wide. After germination started, acorns with radicles 2-4 cm. long were placed on squares of 1/4-inch mesh hardware cloth on top of wide-mouth quart jars capped with aluminum foil. The radicles projected through the hardware cloth and aluminum foil, and extended into Hoagland's nutrient solution with or without the addition of fenuron. The nutrient solutions were aerated continuously. The acorns were covered with a pad of absorbent packing paper kept moist with distilled water. The nutrient solutions contained 0, 5, and 25 p.p.m. fenuron.[4] There were seven seedlings for each treatment.

Root lengths were measured initially and then at intervals during the experiment. Root growth was calculated by difference. The initial heights of the shoots were recorded as zero, since the primary root grows to a considerable length before the shoot emerges.

Injury to the shoots was estimated as percent leaf injury. The development of new growth flushes was observed, and injury to each flush was recorded separately.

Results

Shoot growth was not inhibited for the first 71 days by 5 and 25 p.p.m. fenuron, whereas root growth was markedly inhibited by 25 p.p.m. fenuron. The 5 p.p.m. solution of fenuron affected root growth much less severely

than the 25 p.p.m. solution during the first 50 days, after which root growth gradually ceased as leaf injury increased in severity. Root growth inhibition by 25 p.p.m. fenuron began before shoots emerged and, therefore, before the appearance of visible damage to the leaves. Although root growth was retarded by 25 p.p.m. fenuron, the roots continued to grow for 24 days after the appearance of leaf injury and while the injury increased in severity, which indicates that leaf injury occurred before the energy supply of the seedlings was exhausted. After 29 days, some of the plants treated with 25 p.p.m. fenuron developed moderate to severe leaf injury despite the fact that the food reserves of the acorns were not exhausted, as indicated by a positive starch iodine test. In an attempt to prevent injury to other seedlings, acorns of some uninjured seedlings were bathed in 5 percent glucose solution. The original sets of leaves of the glucose-fed plants and their controls were killed at about the same rate, however. Although feeding glucose through the acorns of fenuron-treated seedlings did not prevent the development of leaf injury, it prolonged the life of the seedlings by supplying energy for additional flushes of growth, all of which became injured and died.

Leaf-feeding of glucose also was attempted as a method of preventing visible injury by fenuron. Untreated leaves of both glucose leaf-fed and distilled water control plants were killed, and injury to both progressed at the same rate.

In another experiment, injury to young seedlings with and without acorns was compared. Excision of the acorns of control seedlings did not reduce root growth; the shoots were capable of supplying adequate nourishment for growth. But excision of the acorns of fenuron-treated seedlings reduced root growth drastically. Leaves of the initial flush of growth of fenuron-treated seedlings with or without acorns were killed. Acorns prolonged the life of fenuron-treated seedlings by providing foodstuff for new flushes of growth but did not prevent leaf injury, whereas fenuron-treated seedlings without acorns failed to produce new growth.

Discussion

The hypothesis on which this study was based is that visible leaf injury resulting from fenuron treatments is due directly to the depletion of the energy supply of the plant as a result of inhibited photosynthesis. This hypothesis is rejected on the basis of the results presented. An alternative hypothesis is that, as a result of a blocked reaction in the photosynthetic mechanism, a product accumulates that is phytotoxic, and this toxic accumulation product is directly responsible for injury to the leaves. Although leaf injury appears to be due to this toxic accumulation product, the ultimate cause of death of the entire plant is starvation.

In addition to the effect of fenuron on the photosynthetic mechanism, a second type of inhibition appears to occur at high fenuron concentrations. This is evident in the reduction of root growth before the emergence of shoots and the development of visible injury to the leaves.

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[1]*Research reported here was conducted while author was employed by Crops Research Division, Agricultural Research Service, U. S. Department of Agriculture. Mention of trade names and commercial enterprises or products is solely for necessary information. No endorsement by the U. S. Department of Agriculture is implied.*

[2]*Plant Physiologist, located at Tempe, in cooperation with Arizona State University; central headquarters maintained at Fort Collins, in cooperation with Colorado State University.*

[3]*Names and dates in parentheses refer to Literature Cited, page 2.*

[4]*Prepared from 80 percent wettable powder formulation of fenuron, supplied by E. I. du Pont de Nemours & Co., Wilmington, Del. Fenuron tested on an active ingredient basis.*

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